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Effects of Sulphur-Containing Compounds on Plasma Redox Status in Muscle-Damaging Exercise

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Abstract

The aim of the study was to compare effects of three-day N-acetylcysteine, α-lipoic acid or taurine administration on plasma antioxidant status and oxidative damage markers in healthy men after performing muscle-damaging exercise. Fifty-five healthy and trained men were randomly assigned to N-acetylcysteine (NAC, 1.8 g/day, 3 days), α-lipoic acid (ALA, 1.2 g/day, 3 days), taurine (TAU, 3 g/day, 3 days) and control group (CON), and exposed to intense resistance exercise. The resistance exercise induced the muscle damage which was observed by significant increase in total creatine kinase (CK) activity at 24 h rest. The administration of NAC and ALA significantly elevated the resting or/and post-exercise plasma total antioxidant status (TAS) and total thiols (TT). Uric acid (UA) concentration was decreased by NAC, ALA and TAU at 24 h rest compared with CON. The plasma lipid peroxidation (TBARS) and protein carbonylation (PC) were considerably reduced by NAC and ALA administration at rest and after exercise. TAU did not have any influence on TAS, TT, TBARS and PC levels. Our study has shown that three-day oral N-acetylcysteine and α-lipoic acid administration enhanced plasma total antioxidant status and attenuated oxidative damage whereas taurine did not demonstrate any antioxidant action in healthy men after performing a single muscle-damaging exercise.

Key Words: oxidative stress, N-acetylcysteine, α-lipoic acid, taurine, physical exercise

Introduction

Studies have shown that reactive oxygen (ROS) and nitrogen species (RNS) are physiological products of aerobic metabolism and are used by cells for a variety of metabolic tasks such as gene expression, protein turnover, inflammatory reaction and erythropoiesis. However, excess ROS and RNS as well as low antioxidant capacity can lead to adverse effects such as oxidative damage which can impair cell metabolism (22, 24). Therefore, some compounds such as cysteine derivatives have been used to aid the antioxidant regeneration and to counteract redox disturbances which can take place during intense physical exercise (7, 13, 14, 17, 18, 23).

N-acetylcysteine, α-lipoic acid and taurine have been taken in sport as a dietary supplements. However, their usage by athletes has given rise to controversy. It has been demonstrated that cysteine derivative supplementation prevents the decline of other antioxidants such as glutathione and antioxidant vitamins, improves glucose metabolism as well as weakens exercise-induced oxidative damages in various tissues (19). On the other hand, it has been shown that N-acetylcysteine or α-lipoic acid administration can lead to temporary enhancement of lipid peroxidation, mitochondrial damage and inhibition of glycogen synthesis (5, 6, 19).

Due to the interesting role of sulphur-containing compounds in cell metabolism and contradictory information concerning their action, the aim of their study was to compare the effects of the short-term

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Table 1. Characteristics of the subjects: control group (CON) and experimental groups (NAC, ALA, TAU)

Group	Age (years)	Height (cm)	Body mass (kg)	Body fat (%)	Lactate (mM)	
					Pre-exercise	Post-exercise
CON n = 15	21.5 ± 1.4	181.7 ± 8.3	87.2 ± 10.6	14.4 ± 4.6	1.40 ± 0.33	8.54 ± 1.78**
NAC n = 15	21.9 ± 1.7	180.7 ± 7.4	87.1 ± 12.8	14.5 ± 5.6	1.61 ± 0.39	8.57 ± 1.92**
ALA n = 10	22.8 ± 1.0	183.7 ± 7.9	86.9 ± 14.0	14.1 ± 4.9	1.89 ± 0.55	8.77 ± 3.30**
TAU n = 15	21.9 ± 1.3	177.1 ± 7.6	82.1 ± 10.7	12.3 ± 5.2	1.76 ± 0.43	8.03 ± 1.67**

*$P < 0.05$ and **$P < 0.01$ significantly different from the pre-exercise value.

administration of N-acetylcysteine, α-lipoic acid or taurine on plasma antioxidant status and oxidative damage markers in healthy men performing muscle-damaging exercise.

Materials and Methods

Subject

Fifty-five healthy trained young males (canoeists and rowers, Table 1) and physical education students participated in this randomized and placebo-controlled study. Subjects did not use any antioxidants supplements (vitamins or minerals) for 4 weeks prior to the study. Subjects were not allowed to exercise for 48 h before and 24 h after the exercise test. All the subjects were informed of the aim of the study and gave their written consent for participation in the research project. The protocol of the study was approved by the local ethics committee in accordance with the Helsinki Declaration.

The subjects were administered for three days the following sulphur-containing compounds: 1.8 g/day of N-acetylcysteine (NAC group; Hexal AG, Holzkirchen, Germany), 1.2 g/day of α-lipoic acid (ALA group; Wörwag Pharma, Böblingen, Germany), 3 g/day of taurine (TAU group; Olimp Sport Nutrition, Warsaw, Poland) or placebo 0.35 g/day of lactose (CON group). The compounds were given in three doses as powder dissolved in 50 ml water. The subjects took the first dose in the morning in a fasted state, the second dose at noon and the last dose 2 h before an evening training.

Exercise Testing

The exercise protocol consisted of three exercises performed in a circuit fashion that included shoulder press, bench press and dead-lift without any breaks. Each exercise element was performed five-times with a constant velocity in one series. After a 1-min break, the load was elevated and then the series was repeated. The subjects performed from 5 to 7 of the series. The initial load was 50% 1 RM (Repetitive Maximal) and then was increased by 10 kg after each series of exercise elements. The 120-kg weight lifted by athletes during training was determined as the maximal load *i.e.* 1 RM. Subjects performed the multi-joint resistance exercise until exhaustion and finishing with a maximal load of 92% 1 RM. The lactate (LA) concentration in the capillary blood was assessed before and immediately after the exercise using LKM 140 Dr Lange kit (Düsseldorf, Germany).

Blood Collection

Blood samples were obtained from a cubital vein with an anticoagulant ($EDTAK_2$) before exercise, immediately after completing exercise and after 24 h of rest. The samples were immediately kept at 4°C after collection. Within 10 min, the blood samples were centrifuged at 2,500 g and 4°C for 10 min. Aliquots of plasma were stored at -20°C. All the samples were analyzed within 7 days.

Biochemical Measurements

Plasma total creatine kinase (CK) activity was evaluated using the diagnostic assay for the kinetic enzyme analyzer Konelap 60 BioMerieux (Craponne, France). The CK detection limit was 6 U/ l. The intra-assay coefficient of variation (CV) for the CK kit was 1.85%.

Total antioxidant status (TAS) of the plasma was measured using the method developed by Randox Laboratories Ltd. (Warsaw, Poland). The method is based on the formation of 2'-2'-azino-di-[3-ethylbenz-thiazoline sulphonate] radical which is measured

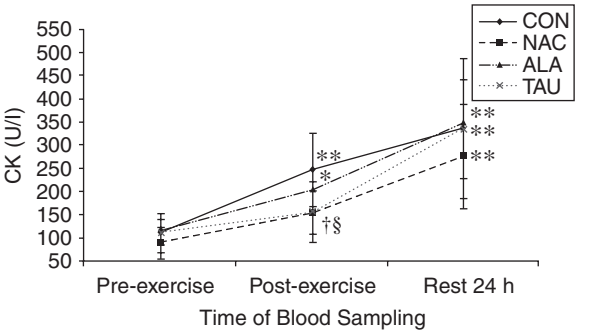

Fig. 1. Changes in total activities of creatine kinase (CK); *P < 0.05 and **P < 0.01 indicate post-exercise *vs*. pre-exercise values; †P < 0.01 indicates CON *vs*. NAC; §P < 0.05 indicates CON *vs*. TAU.

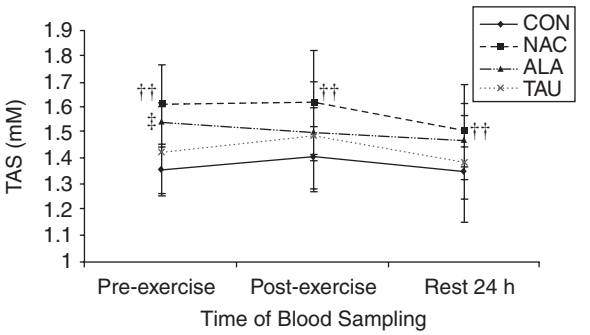

Fig. 2. Changes in total antioxidant status (TAS); ††P < 0.01 indicates CON *vs*. NAC; ‡P < 0.05 indicates CON *vs*. ALA.

spectrophotometrically at 600 nm. The detection limit for the TAS kit was 0.21 mM and the intra-assay CV was 2.77%.

Plasma total thiols (TT) concentration was estimated by the method of Hebeeb (11) using dithionitrobenzene (DTNB; Sigma, Poznan, Poland). The samples were added to a denaturing solution containing sodium dodecyl sulfate to ionise the sulfhydryl groups in order to make them more reactive to DTNB. The samples were measured at 410 nm against a control sample (minus DTNB). The intra-assay CV for the thiol procedures was <10%.

Plasma uric acid (UA) concentration was measured using a commercial kit (Emapol, Gdansk, Poland) based on the uricase-peroxidase system. The UA detection limit for the procedure was 10 μM and the intra-assay CV was 1.85%.

Plasma lipid peroxidation products were estimated using the measurement of thiobarbituric acid (Fluka, Poznan, Poland)-reactive substance (TBARS) level according to the method of Buege and Aust (4). To avoid further peroxidation, plasma samples were treated with 15% trichloroacetic acid (POCH, Gliwice, Poland) containing 0.25 M hydrochloric acid (POCH, Gliwice, Poland) immediately after centrifugation. The TBARS level was expressed as nmol of malondialdehyde using 1,1,3,3- tetraethoxypropane as a standard (Fluka, Poznan, Poland). TBARS detection limit for the method was 0.13 nmol/ml. The intra-assay CV for the TBARS procedure was <10%.

Plasma protein carbonyls (PC) were measured by the method of Levine *et al*. (15) using 2,4-dinitrophenyl hydrazine (Fluka, Poznan, Poland). The carbonyl content was calculated using a molar extinction coefficient of 22,000 M/cm and expressed as nmol PC per mg of plasma protein. The carbonyl content was expressed as nmol PC per mg of plasma protein. Protein concentration was estimated by the Bradford method (3) standardised with bovine serum albumin (POCH, Gliwice, Poland).

Statistics

Statistical analysis was carried out using Statistica 8.0. (StatSoft, Krakow, Poland). All the data were tested for their normal distribution. To determine the effects of exercises and supplements as well as the effects of interaction between exercise and supplementation, statistical analysis was performed using the two-way ANOVA and the *post-hoc* Tukey's test. The accepted level of significance was defined as P < 0.05. Results are expressed as means ± SD.

Results

The resistance exercise protocol caused the muscle damage in subjects which were demonstrated through statistically significant increase in total activity of CK at 24 h after exercise (Fig. 1). CK release from skeletal muscle was significantly reduced immediately after exercise by NAC and TAU administration. CK did not correlate with any markers of oxidative damage.

Even though, the resistance exercise did not induce any changes in TAS, administration with NAC and ALA significantly elevated TAS before exercise and during recovery. TAU did not demonstrate any effect on antioxidant status (Fig. 2). TT concentration increased in response to exercise in each group. NAC or ALA intake additionally elevated the resting total thiols concentration whereas TAU did not affect TT levels (Fig. 3). In the CON group, the UA concentration increased by 24% at 24 h of rest. The use of sulphur-containing compounds, mainly NAC and TAU, prevented the changes in UA following the resistance exercise (Fig. 4).

The TBARS concentration significantly increased immediately after exercise in all the subjects whereas PC concentration did not respond to resis-

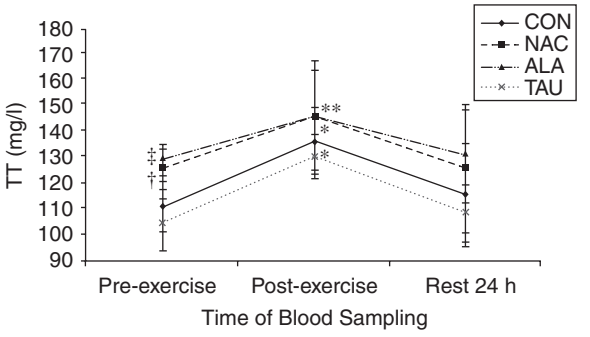

Fig. 3. Changes in total thiols (TT) concentration; *$P < 0.05$ and **$P < 0.01$ indicate post-exercise *vs*. pre-exercise values; $^{\dagger}P < 0.05$ indicates CON *vs*. NAC; $^{\ddagger}P < 0.05$ indicates CON *vs*. ALA.

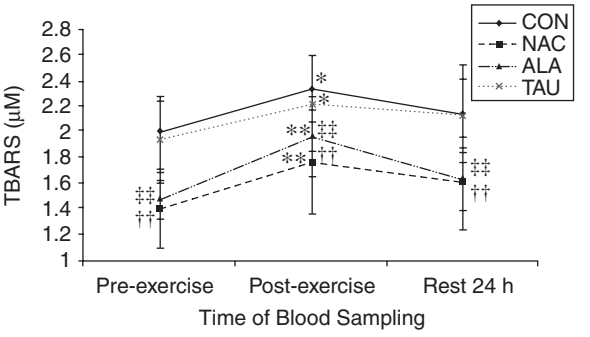

Fig. 5. Changes in lipid peroxidation (TBARS) concentration; *$P < 0.05$ and **$P < 0.01$ indicate post-exercise *vs*. pre-exercise values; $^{\dagger\dagger}P < 0.01$ indicates CON *vs*. NAC; $^{\ddagger\ddagger}P < 0.01$ indicates CON *vs*. ALA.

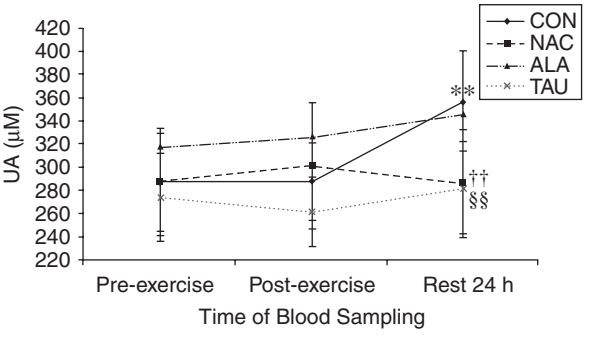

Fig. 4. Changes in uric acid (UA) concentration; **$P < 0.01$ indicates post-exercise *vs*. pre-exercise values; $^{\dagger\dagger}P < 0.01$ indicates CON *vs*. NAC; $^{\S\S}P < 0.01$ indicates CON *vs*. TAU.

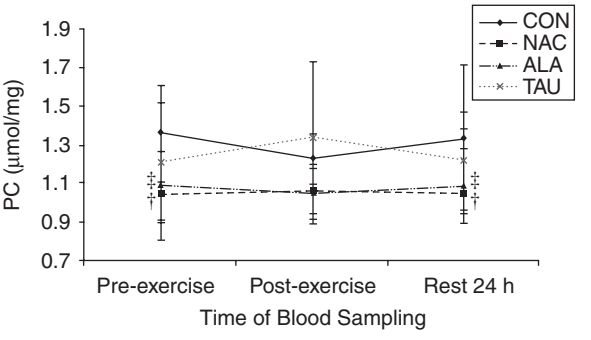

Fig. 6. Changes in protein carbonylation (PC) concentration; $^{\dagger}P < 0.05$ indicates CON *vs*. NAC; $^{\ddagger}P < 0.05$ indicates CON *vs*. ALA.

tance exercise (Figs. 5-6). NAC and ALA revealed a distinct antioxidant action in relation to plasma lipid peroxidation and protein carbonylation. Both supplements caused over 16% decline in TBARS and PC concentrations before exercise and during recovery. TAU administration did not demonstrate any antioxidant action that is TBARS and PC did not change in the TAU group compared with the CON group.

Discussion

By measuring the total capability of plasma antioxidants to reduce ROS and RNS, it has been found that their contributions were 35-65% from urate, 0-24% from ascorbate, 5-10% from vitamin E and 10-50% from plasma proteins (27). The plasma total antioxidant status (TAS) has been relatively static parameter which depends on many factors such as intensities of cell ROS and RNS generation, concentrations of stress hormones and antioxidants, cysteine availability and glutathione synthesis. Therefore, it has been difficulty to observed the distinct shift in total antioxidant status following a single intense exercise. The present study has not shown any effect of exercise on TAS, similarly to others (6, 23). Nevertheless, NAC and ALA administration significantly elevated total antioxidant status in plasma. This could be related to fast and active transport of NAC into cells, deacylation and use of cysteine in glutathione synthesis and then the releasing of glutathione or cysteine from the cells. The study of Nielsen *et al*. (21) has demonstrated a high cysteine level in plasma after three-day NAC supplementation (6 g/day) in athletes. Medved *et al*. (17) have shown that pre-exercise N-acetylcysteine infusion increases blood glutathione in sprint tests and recovery. Contrary to NAC, ALA has been an autonomous element of plasma antioxidant status, not as a substrate to glutathione synthesis. ALA can increase thiol concentration only through reduction of disulphide glutathione and improvement of cystine utilization (9).

In resting, NAC and ALA administration significantly increased TT concentration whereas TAU did not induce any changes in TT and TAS. The lack of effect of TAU on the antioxidant status is contrary to previous results. Dawson *et al*. (7) and Zhang *et al*.

(30) have maintained that taurine markedly influences concentration of cysteine derivatives. Miyazaki *et al*. (18) have shown that 2-week taurine administration in rats with 100 and 500 mg/kg/day affect glutathione level inhibiting its oxidation.

Uric acid, like thiols, has been an important element of plasma antioxidant status. UA acts through the formation of stable coordination complexes with iron ions and prevents Fenton's reaction. Moreover, UA is able to reduce ROS and stabilize other plasma antioxidants such as thiols and ascorbic acid (25). The antioxidant properties of UA were confirmed by Waring *et al*. (26) in volunteers who took a uric acid solution. UA solution raised the serum urate concentration, accompanied by increases in antioxidant status and decrease in circulating oxidative stress markers 8-iso-prostaglandin F2α. On the other hand, UA is a final product of xanthine oxidase reaction which generates reactive oxygen species during physical exercise (25). The increase in UA concentrations might be a physiological or adaptive response to exercise to augment the antioxidant mechanisms (2). Our study has shown that resistance exercise causes an increase in plasma UA concentration whereas administration of cysteine derivatives administration prevents changes in UA at 24 h rest and reduces its participation in plasma antioxidant status. Similar results were obtained by Fuller *et al*. (8) who observed the low levels of blood UA and reduction in ischemia/reperfusion injures in kidneys after NAC administration. The mechanism of interaction between UA and thiol levels could be related to effects of thiols, mainly glutathione, on xanthine oxidase and dehydrogenase activity (12).

The detection of lipid peroxidation and protein carbonylation products using thiobarbituric acid and dinitrophenyl hydrazine have been the most widely used markers of oxidative damage. Reduction of peroxidation and carbonylation in various tissues, followed by thiol compounds, were observed by many authors (1, 10, 16, 20, 29). In the present study, NAC or ALA administration markedly lowered the plasma PC and TBARS levels compared with controls, and maintained their antioxidant action for 24 h after administration of the last doses. However, Childs *et al*. (6) and Moini *et al*. (19) obtained the opposite results. The authors observed the temporal increase in lipid peroxidation after NAC or ALA administration. The excess of thiols can cause the thiol auto-oxidation which is a potential source of reactive oxidants and may contribute to the cytotoxicity of reactive thiols such as cysteine and cysteamine (28). It should be stressed that the applied doses of NAC and ALA in this study were optimal and did not induce oxidative damages.

Contrary to NAC and ALA, taurine did not reveal any antioxidant action, *i.e*. TAU did not influence on lipid peroxidation or protein carbonylation. The results concerning TAU have been different from the effects of Dawson *et al*. (7) and Zhang *et al*. (30) who observed significant decreases in peroxidation products concentrations in blood and skeletal muscles after one-month or seven-day TAU supplementation, respectively. The applied dosages of taurine in our study were probably insufficient or the supplementation period was too short in order to affect TBARS and PC. Further studies are necessary to explain this discrepancy and to establish the effective taurine dosage.

In summary, three-day administration with 1.8 g N-acetylcysteine or 1.2 g α-lipoic acid led to improvement of total antioxidant status and confirmed the significant antioxidant action through the reduction in lipid peroxidation protein carbonylation whereas taurine intake at a dose of 3 g daily did not influence plasma pro-antioxidant status in healthy men after performing a single muscle-damaging exercise.

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